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Patterns of host specificity and transmission among parasites of wild primates

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Abstract

Multihost parasites have been implicated in the emergence of new diseases in humans and wildlife, yet little is known about factors that influence the host range of parasites in natural populations. We used a comprehensive data set of 415 micro- and macroparasites reported from 119 wild primate hosts to investigate broad patterns of host specificity. The majority (68%) of primate parasites were reported to infect multiple host species, including animals from multiple families or orders. This pattern corresponds to previous studies of parasites found in humans and domesticated animals. Within three parasite groups (viruses, protozoans and helminths), we examined parasite taxonomy and transmission strategy in relation to measures of host specificity. Relative to other parasite groups, helminths were associated with the greatest levels of host specificity, whereas most viruses were reported to infect hosts from multiple families or orders. Highly significant associations between the degree of host specificity and transmission strategy arose within each parasite group, but not always in the same direction, suggesting that unique constraints influence the host range of parasites within each taxonomic group. Finally characteristics of over 100 parasite species shared between wild primates and humans, including those recognised as emerging in humans, revealed that most of these shared parasites were reported from multiple host orders. Furthermore, nearly all viruses that were reported to infect both humans and non-human primates were classified as emerging in humans.
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1. Introduction

The majority of parasites examined to date, including over 60% of human pathogens and 80% of those infecting domesticated animals, are capable of infecting multiple host species (Cleaveland et al., 2001; Taylor et al., 2001; Woolhouse et al., 2001). Recent emerging diseases in humans, including those caused by Ebola, Influenza A, Nipah, and SARS viruses, are associated with multihost pathogens maintained in non-human animal populations (Murphy, 1998; Hahn et al., 2000; Dobson and Foufopoulos, 2001; Taylor et al., 2001; Williams et al., 2002). Multihost pathogens also pose problems for wildlife conservation, and have caused population declines or high mortality among African carnivores, harbor seals, sea otters, black-footed ferrets, and African apes (Roelke-Parker et al., 1996; Harvell et al., 1999; Daszak et al., 2000; Jensen et al., 2002; Miller et al., 2002; Walsh et al., 2003). Despite their threats to global health and conservation, factors that influence the evolution and ecology of multihost parasites and their distributions in wild animal populations are not well understood (Desdevises et al., 2002), in part because conventional studies have focused most commonly on single host-pathogen systems (Anderson and May, 1991;

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Bull, 1994; Day, 2001) or on domesticated animals and humans (Cleaveland et al., 2001; Taylor et al., 2001).

A variety of ecological and evolutionary factors may influence patterns of parasite specificity. Here we focus on two key variables. First, transmission strategies that increase encounters with new hosts, such as transmission by biting arthropods or through contaminated soil or water, are predicted to increase the range of hosts that a pathogen can infect (Woolhouse et al., 2001). Second, greater genetic variability and more rapid generation times might allow certain pathogens to readily exploit new host species. For instance, parasites with high antigenic variation should have an increased ability to recognise host proteins or evade host immune defenses (Bitter et al., 1998; Simon et al., 1998).

In this study, we assembled a comprehensive database of infectious diseases reported from wild primate populations to investigate the frequency and characteristics of multihost pathogens. Indeed, several recent papers have highlighted that a majority of parasites reported to infect humans and domesticated animals can in fact infect more than one host species (Cleaveland et al., 2001; Taylor et al., 2001). This is not necessarily surprising because both humans and domesticated animals have cosmopolitan distributions, exist in large dense populations, and can be exposed to a wide diversity of pathogens from wildlife sources. In contrast to these earlier papers, our study focused on parasites reported from wild populations of non-human primates, in part because they represent a diverse group of mammals that have been threatened by infectious diseases (Wallis and Lee, 1999; Walsh et al., 2003; Leroy et al., 2004), and they have been identified as wild reservoirs or ancestral hosts for a growing number of human pathogens (Wolfe et al., 1998, 2004).

Using a similar database of host-pathogen records, Nunn et al. (2003) tested a suite of socioecological factors predicted to influence parasite species richness in wild primates, and demonstrated that host population density and geographic range size were positively associated with parasite species richness in wild primates. In this paper, we focused on traits of the parasites themselves by examining the association between host specificity and transmission strategy within and among multiple parasite taxonomic groups. We predicted that parasites transmitted by direct contact would be relatively more host specific than parasites with greater opportunities for among-host dispersal. We also predicted that viruses, owing to their short generation times and high mutation rates, would have broader host ranges. Finally we investigated the transmission and specificity of parasites reported from both humans and wild non-human primates to ascertain the risks that these shared parasites pose in relation to human emerging diseases.

2. Materials and methods

2.1. Parasite records from wild primates

A database of disease-causing organisms reported from wild primate populations was compiled from 258 published references, including scientific papers and book sections. We searched for reports using primate species' Latin binomials as keywords in major online reference databases (Biological Abstracts, AGRICOLA, Medline, Web of Science). We also searched by primate genus name, following the taxonomic scheme of Corbet and Hill (1991), as well as by common taxonomic variants (based on Rowe, 1996; Groves, 2001). The vast majority of papers and reports used in our data set were published between 1970 and 1999, with the total time frame spanning 1940–2002. For each parasite species reported from a wild primate population, we recorded information on hosts, parasites and sampling locality. We defined parasites broadly to include viruses, protozoa, fungi, arthropods, helminths and bacteria. Our original data set included 2173 cumulative lines of data, where each line was a record of a parasite species reported from a wild primate population. Nomenclature for viruses followed the International Committee on the Taxonomy of Viruses database (ITCVdb) available online (http://www.ncbi.nlm.nih.gov/ICTVdb/Ictv/), and nomenclature for other parasite species followed the guidelines published by the National Center for Biotechnology Information (NCBI). Additional details about the database are described in Nunn et al. (2003), Nunn and Altizer (2005), and are available on the Global Mammal Parasite Database website (www.mammalparasites.org). From our original host-parasite database, we generated a list of parasite species reported from free-living primate populations. To this initial list of parasite species, we recorded whether or not the parasite has been reported to infect humans based on records from Ashford and Crewe (1998) and Taylor et al. (2001), and also recorded information on transmission and specificity described below.

2.2. Scoring parasite specificity

For all parasite species, information on the taxonomic range of primates and other species reported as hosts was compiled by searching for published records of vertebrate animals infected under natural conditions using two online databases (BIOSIS and Web of Science). To assess virus specificity, we searched the PubMed online database (in addition to Web of Science) using ICTV virus names. We assigned a score from 1 to 5 to each parasite based on the taxonomic affiliation of affected hosts as follows: 1 = species specific; 2 = genus specific; 3 = family specific; 4 = order specific; and 5 = vertebrate specific (i.e. can infect multiple host orders). For host groupings at the level of primate families, we followed the taxonomy of Corbet and Hill (1991). We based specificity scores on recorded

infections from wild populations only (i.e. excluding records of parasites from captive animals or infection experiments), and considered only vertebrate host species in assigning specificity scores, ignoring invertebrates that might serve as intermediate hosts or vectors.

To each specificity score, we added a confidence score based on the quality and quantity of published records that provided information on affected host species. Highest confidence scores (3) were assigned to records based on direct isolation of pathogens or their DNA, in combination with a sufficient number of published records (usually 5 or more); intermediate confidence (2) was assigned to scores based on indirect measures of pathogen presence (e.g. serology) or a limited number of published records, and low confidence (1) was assigned to specificity scores based on unpublished knowledge, strong inference, or information derived solely from our primate parasite database. Confidence scores were used to evaluate the sensitivity of patterns by analyzing the data once with all available records, and second by excluding data with low confidence. All viral specificity scores were assigned high or moderate confidence; thus, for this group we did not repeat analyses excluding low confidence data.

As a second measure of confidence, we examined the sampling effort associated with each parasite species using citation counts from three common bibliographic databases: ISI Web of Science (WOS; www.isiwebofknowledge.com), Biosis Previews (www.biosis.org), and Pubmed (www.pubmed.org). We searched each database for corrected parasite Latin binomials or ICTVdb virus names. For each parasite species we computed average sampling effort as the log-transformed arithmetic mean of all three citation counts (adding one to each count first to avoid taking the log of zero). Analysis of variance showed that categorical measures of confidence were highly associated with the average of citation counts ($F_{2,409}=132.9$; $P<0.0001$) so that specificity scores assigned high confidence were also generally from the best studied parasites in the scientific literature (mean log-references = 2.18). Parasites with moderate confidence scores had intermediate numbers of citations (mean log-references = 1.04), and those with the lowest confidence were generally not well represented in the scientific literature (mean log-references 0.29).

Not surprisingly, specificity scores were associated with both categorical and continuous measures of confidence. In particular, the least specific parasites were associated with the highest confidence scores, whereas genus or species specific parasites were more likely to be assigned intermediate or low confidence scores. This association was highly significant when examined across all parasite groups ($\chi^2=193.6$, d.f. = 8, $P<0.0001$), as well as within each of the three major parasite groups: viruses ($\chi^2=15.3$, d.f. = 4, $P=0.004$), protozoa ($\chi^2=33.3$, d.f. = 8, $P<0.0001$), and helminths ($\chi^2=61.5$, d.f. = 8, $P<0.0001$). A similar pattern was found when we examined the association between specificity and average citation counts ($F_{4,407}=83.04$; $P<0.0001$). In this case, mean citation counts were lowest among parasites assigned genus- or species-specific status (mean log-references = 0.25 and 0.34, respectively). Citation counts were intermediate for parasites assigned family specific status (mean = 0.90) and were greatest for parasites in the order-specific and multi-order categories (mean = 1.83 and 2.24, respectively).

2.3. Parasite transmission mode

The transmission strategy of each parasite species was recorded as one or more of the following four categories: close contact, non-close contact, biting arthropod vectors, or intermediate hosts. Parasites spread by close contact were those that were highly contagious and communicable by close proximity or direct contact such as biting, scratching, mating contact, or other touching. Close contact transmission was further subdivided into three routes—sexual, vertical (parent to offspring), or close non-sexual contact. Non-close contact involved transmission via fomites or contact with contaminated soil or water. Vector-borne parasites were those spread via biting arthropods (ticks, mites, fleas, flies, and other invertebrates), and parasites with intermediate hosts were those characterised by the presence of complex life cycles and/or trophic transmission. Transmission strategies were assigned by multiple web searches using parasite Latin binomials or ICTV virus names. If direct information was not available for a particular parasite species, transmission strategies were assigned based on information from closely related parasites (if similar transmission strategies were highly likely) or remained unassigned.

2.4. Data analysis

We first examined the occurrence of host specificity and major transmission strategies across all parasites from wild primates, and then within each of the three best-represented groups: viruses, helminths, and protozoa. Second, we tested whether the predicted associations between transmission and host specificity were consistent with observed patterns within each of the major parasite groups. Finally, the taxonomic distribution of parasites from wild primates (arthropods, bacteria, fungi, helminths, protozoa, and viruses) was compared with reports from earlier studies of humans and domesticated animals (e.g. Cleaveland et al., 2001; Taylor et al., 2001) to investigate whether the types and characteristics of parasites from wild hosts were similar to those reported for economically important species, and to examine characteristics of parasites from wild primates that were also reported as emerging in human populations. Unless otherwise stated, we used Pearson Chi square analysis or logistic regression (general linear model with binomial errors) to examine associations between host specificity, parasite taxonomy, and transmission strategy.

3. Results

A total of 415 parasite species from 119 host species were represented in the database of parasites reported from wild primate populations. The distribution of parasite species was: 163 helminths (39%), 82 protozoans (20%), 82 viruses (20%), 46 arthropods (11%), 32 bacteria (8%) and 10 fungi (2%). Thus, three major groups—helminths, protozoa, and viruses—collectively represented 79% of all parasites reported from wild primates.

3.1. Patterns of host specificity

Of all primate parasites, the majority (68%) were reported to infect multiple host species, and only 131 (32%) were identified as species specific. Among multi-host parasites, 40% were reported from both primate and non-primate hosts, and another 25% were reported from multiple primate families ($n=284$). Among the less well represented taxonomic groups, multihost pathogens were common—70% of all arthropods, 90% of bacteria, and 90% of fungi were reported to infect more than one host species, and in most cases affected hosts were from multiple orders.

Host specificity varied dramatically among the three best-represented groups of primate parasites (Fig. 1). Viruses were dominated by multi-order parasites (Fig. 1(a)); only 13% were categorised as species specific, and nearly half of all primate viruses were also reported to infect non-primate hosts. Protozoan parasites were nearly evenly distributed among all five levels of specificity (Fig. 1(b)). Most helminths, on the other hand, were categorised as host specific, with 48% recorded from single host species (Fig. 1(c)). These differences among the three major parasite groups were statistically significant when tested using all parasite records ($\chi^2=76.0$, d.f.$=8$, $P<0.0001$), and remained significant when we removed data from parasites with low confidence scores ($\chi^2=31.7$, d.f.$=8$, $P<0.0001$) or those with five or fewer references on average ($\chi^2=44.22$, d.f.$=8$, $P<0.0001$).

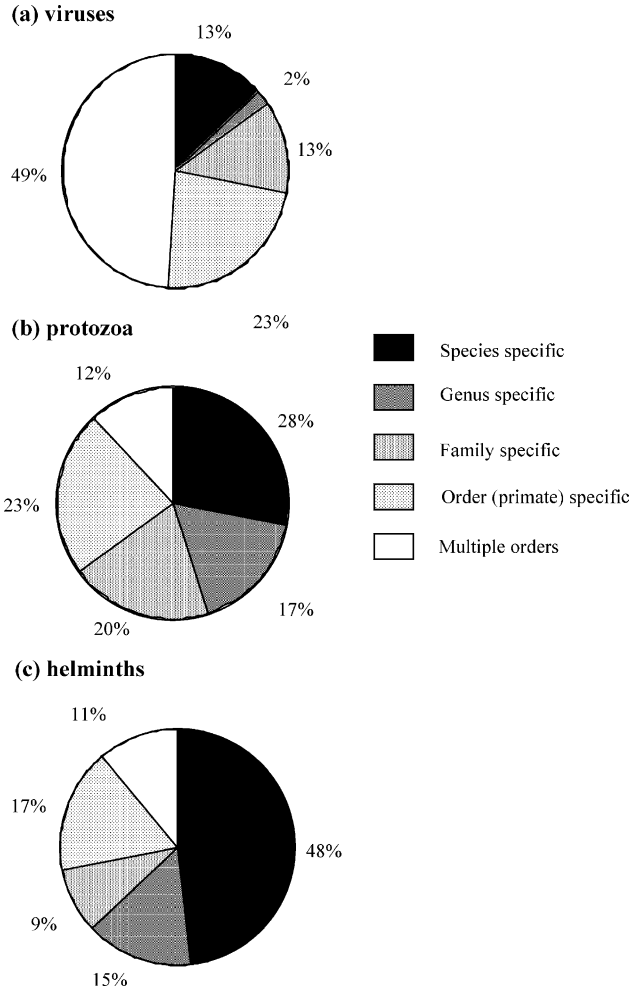

Fig. 1. Levels of host specificity among the three best-represented parasite groups from wild primates: (a) viruses ($n=82$), (b) protozoa ($n=82$), and (c) helminths ($n=163$). Differences in host specificity levels were significantly different among the three parasite groups shown.

3.2. Transmission strategies

Of the 408 parasite species for which we could identify major transmission routes, 185 (45%) could be transmitted by close non-sexual contact, 20 (5%) by sexual contact, 20 (5%) by vertical transmission, 175 (43%) by non-close contact, 132 (32%) by arthropod vectors, and 59 (14%) through intermediate hosts or trophic transmission. These categories were not mutually exclusive, with 34% (139) of all parasite species being associated with more than one transmission strategy (and, therefore, counted more than once in the above percentages).

The importance of different transmission routes varied significantly among the three major parasite groups (Fig. 2). Close contact transmission (including sexual, non-sexual and vertical routes) was most common among viruses (Fig. 2(a)) and was less common among helminths and protozoa ($\chi^2=33.96$, d.f.$=2$, $P<0.0001$). In fact, viruses were the only pathogen group where sexual transmission was relatively common, with 17 (20%) of all primate viruses transmitted by sexual contact. Moreover, there was a high degree of overlap among sexual, vertical, and close non-sexual transmission, in that 35% of primate viruses transmitted by close non-sexual contact could also be transmitted both sexually and vertically. Vector transmission, on the other hand, was far more common among protozoa than other parasite groups (Fig. 2(b); $\chi^2=35.0$, d.f.$=2$, $P<0.0001$). Finally, helminths were the only group where transmission by intermediate hosts was common, with another 25% of helminths transmitted by biting vectors (Fig. 2(c)).

3.3. Associations between host specificity and transmission mode

Across all parasites we found a significant association between transmission mode and specificity score ($\chi^2=27.43$,

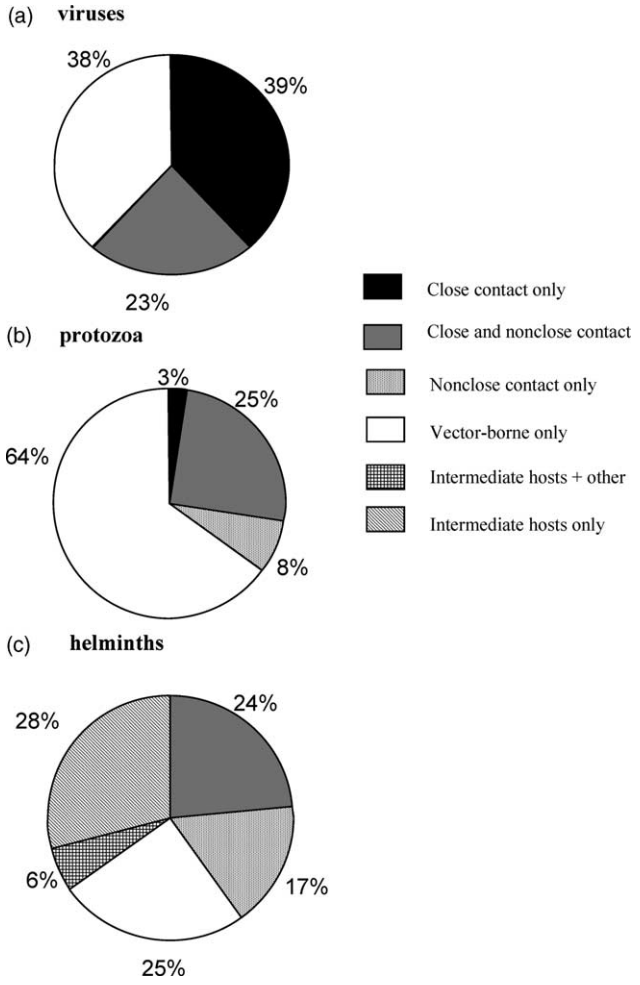

Fig. 2. Transmission strategies among the three best-represented parasite groups from wild primates: (a) viruses ($n=81$), (b) protozoa ($n=80$), and (c) helminths ($n=157$). Close contact here includes close non-sexual contact in addition to vertical and sexual transmission (although these latter two modes are represented only by viruses). Because some parasites could be transmitted by more than one strategy, we show the frequencies of parasites with both single and combined strategies.

d.f. = 16, $P=0.037$; Med/High confidence: $\chi^2=20.35$, d.f. = 16, $P=0.205$), but visual inspection of the data indicated that different taxonomic groups exhibited contrasting patterns. Therefore, we investigated the relationship between host specificity and transmission within viruses, protozoa and helminths in two ways. First, we assigned each parasite species a single transmission strategy based on the singular and combined modes shown in Fig. 2 (close contact, non-close contact, close + non-close contact, vector, and intermediate host). Second, we examined each route alone and contrasted parasites capable of transmission by that route against all other parasites.

3.3.1. Viruses

Associations between categorical measures of viral transmission and specificity were highly significant ($n=80$; $\chi^2=39.35$, d.f. = 8, $P<0.0001$). Approximately half of all viruses transmitted by close contact, or by a combination of close and non-close contact, were specific at the level of primate species, genus or family (Fig. 3(a); Table 1a). As predicted, parasites transmitted by sexual or vertical routes tended to be highly host specific ($\chi^2_{\text{sexual}}=36.51$, d.f. = 4, $P<0.0001$, $\chi^2_{\text{vertical}}=48.22$; d.f. = 4; $P<0.0001$). In contrast, nearly all vector-borne viruses were reported from hosts from multiple orders (Fig. 3(a)) and this result was highly significant (Table 1(a)). All but one of these vector-borne viruses were RNA viruses. Furthermore, we observed a major dichotomy in specificity patterns among DNA vs. RNA viruses, in that virtually all (86% of 57) RNA viruses in our database could infect hosts from multiple families or orders (with the exception being nine retroviruses), whereas specificity levels among the 25 DNA viruses were more evenly distributed among specificity categories.

3.3.2. Protozoa

Protozoa were dominated by vector-borne parasites but also included a large number of host-specific parasites. As with viruses, we found highly significant associations between specificity scores and categorical transmission strategies of protozoan parasites using all data ($\chi^2=29.27$, d.f. = 12, $P=0.007$), and when excluding specificity scores with low confidence ($\chi^2=28.76$, d.f. = 12, $P=0.010$) or for which five or fewer citations on average were reported ($\chi^2=21.28$, d.f. = 12, $P=0.026$). In contrast to the patterns found in viruses, however, vector borne protozoa were primarily reported from single primate species, genera, or families (Fig. 3(b)), and this positive association between vector transmission and host specificity was highly significant (Table 1(b)). By comparison, the majority of protozoa transmitted by both close and non-close contact were only specific at the level of host order or beyond (Fig. 3(b)), and again, this association was highly significant (Table 1(b)).

3.3.3. Helminths

Helminths were generally specific to single host species, genera, or family (Fig. 3(c)), but levels of specificity differed significantly among the four major transmission strategies when tested using data for all helminths ($\chi^2=41.4$, d.f. = 12, $P<0.0001$), and when restricting the analysis to specificity scores with high or moderate confidence only ($\chi^2=31.20$, d.f. = 12, $P=0.002$)—but results were non-significant when performed using the subset of parasite species for which an average of five or more citations were reported. Helminths capable of transmission by close contact were primarily specific to single host species (Fig. 3(c)) and this association was highly significant (Table 1(c)). Incidentally, all helminths transmitted by close contact could also be transmitted by non-close contact (Fig. 3(c)). Vector-borne helminths also tended to be relatively host specific, and many were reported only from single host species (Fig. 3(c); Table 1(c)).

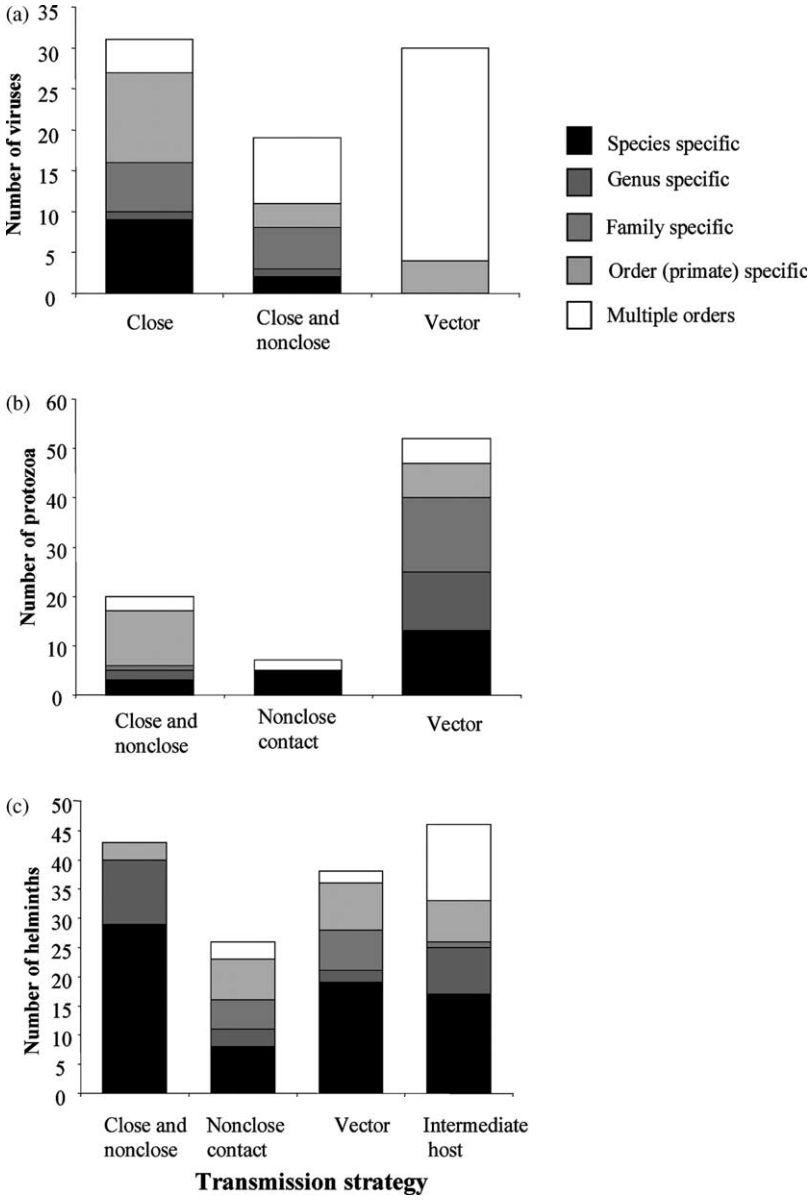

Fig. 3. The relationship between transmission strategy and host specificity within the three best-represented groups: (a) viruses ($n=80$), (b) protozoa ($n=79$), and (c) helminths ($n=153$). The number of parasites transmitted by each strategy is represented by the height of each bar, and the specificity scores assigned to individual parasite species are represented by colors within each bar, with darker colors indicating a greater level of host specificity.

A higher proportion of helminths transmitted by intermediate hosts or non-close contact alone could infect hosts from multiple families or orders (Fig. 3(c) and Table 1(c)).

3.4. Comparison with parasites from humans and domesticated animals

The taxonomic distribution of parasites of wild primates (i.e. non-human primates) differed from parasites reported from humans and domesticated species (Fig. 4(a)–(d); note that arthropods were omitted for this comparison). The most significant differences in the taxonomic representation of parasites occurred between humans vs. wild primates (Fig. 4(a) and (b)), and this difference was highly significant ($\chi^2=318.03$, d.f.$=4$, $P<0.0001$). In particular, 87% of parasites from wild primates were helminths, viruses and protozoa, whereas bacteria and fungi comprised the majority (60%) of human pathogens.

Despite these differences, there was a high degree of overlap among parasites from humans and wild primates, with 114 (27.5%) shared between wild primates and humans (Table 2). Approximately half of all bacteria and fungi reported from wild primates were also reported as human pathogens. Even though the greatest total number of parasites shared between the two databases involved helminths, only 20% of all wild primate helminths were reported to infect humans. As predicted, the vast majority (90%) of non-human primate parasites also reported from

Table 1
Associations between parasite transmission strategy and host specificity based on chi-square analysis of major transmission routes (presence/absence) in relation to host specificity scores

(a) Viruses

Transmission strategy	All data ($n=80$)[a]			
	Dir	χ^2	d.f.	P[b]
Close contact	+	33.24	4	0.000
Close+non-close	NS	4.77	4	0.372
Vector borne	−	31.60	4	0.000

(b) Protozoans

Transmission strategy	All data ($n=79$)				Excl. low confidence ($n=50$)			
	Dir	χ^2	d.f.	P	Dir	χ^2	d.f.	P[c]
Close contact	−	18.44	4	0.001	−	14.05	4	0.007
Non-close contact	+	13.12	4	0.011	+	12.17	4	0.016
Close+non-close	−	16.97	4	0.002	−	14.10	4	0.007
Vector borne	+	16.37	4	0.003	+	14.31	4	0.006

(c) Helminths

Transmission strategy	All data ($n=163$)				Excl. low confidence ($n=80$)			
	Dir	χ^2	d.f.	P	Dir	χ^2	d.f.	P[d]
Close+non-close	+	23.04	4	0.000	+	23.20	4	0.000
Non-close contact	−	15.10	4	0.005	−	12.10	4	0.016
Vector borne	+	10.91	4	0.028	NS	2.56	4	0.633
Intermediate host	−	31.72	4	0.000	−	9.95	4	0.014

Each analysis was repeated using all available data and those for which high or moderate confidence was assigned to specificity scores. Symbols (+/−) indicate the directionality of the association (i.e. whether parasites with that transmission strategy were more or less specific than parasites transmitted by other routes). χ^2, Chi-square scores. d.f., Degrees of freedom, P, P value corrected by the Bonferroni method ($\alpha=0.05/n$, where n is the number of tests and a, b, c and d refer to the Bonferroni corrected P values.

[a] No low confidence scores.
[b] $\alpha=0.017$.
[c] $\alpha=0.0125$.
[d] $\alpha=0.0125$.

humans were recorded as multihost parasites, capable of infecting hosts from multiple families and orders.

Viruses reported from both humans and wild primates were primarily RNA viruses (85% of shared viruses), and over half were transmitted by arthropod vectors (Fig. 5). Protozoa reported to infect both wild primates and humans spanned multiple phyla and were transmitted either by a combination of close and non-close contact (70%) or arthropod vectors (29%; Fig. 5). Approximately half (45%) of the helminths that overlapped with wild primate and human hosts had complex life cycles and transmission by intermediate hosts, and the remainder were nematodes transmitted by non-close contact or arthropod vectors (Fig. 5). Only 10% of the shared parasites were recorded as being transmitted by close contact only, and all of these were viruses.

Finally, 44.7% (51 of 114) of non-human primate parasites reported to infect humans were classified as emerging based on a recent analysis of risk factors associated with human diseases (Taylor et al., 2001). Over half of these 'emerging parasites' were viruses; in fact, 28% of all viruses reported from wild primates were classified as emerging in humans (Table 2). Moreover, there was a significant association between emerging diseases and transmission strategy ($\chi^2=15.37$; d.f.$=4$; $P=0.008$). Given that a shared parasite was transmitted by close contact, it was highly likely to be recorded as emerging in humans (Table 2(b)), and the same was true for parasites transmitted by arthropod vectors. Shared parasites transmitted by intermediate hosts were least likely to be classified as emerging in humans (Table 2(b)).

4. Discussion

We found that a remarkable number of primate pathogens (68%) were reported to infect multiple host species. Since most of these affected hosts were from more than one family or order, our data suggest that species-specific parasites are an exception to the rule (Fig. 1). This observation is consistent with patterns from studies of pathogens in humans and domesticated animals (Cleaveland et al., 2001; Taylor et al., 2001; Woolhouse et al., 2001). However, it runs counter to the hypothesis that host specificity (and, more generally, niche specialisation) offers greater opportunity for diversification and coexistence among parasitic organisms and their hosts (Berenbaum, 1996; McPeek, 1996; Poulin, 1998). A number of factors

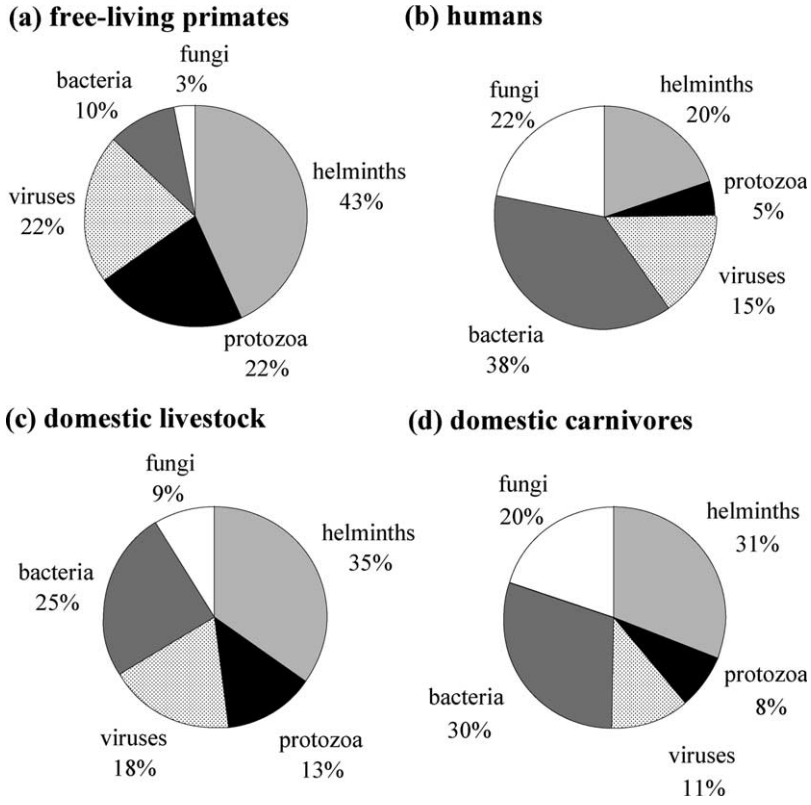

Fig. 4. Taxonomic distribution of parasites from (a) free-living primates ($n=369$), (b) humans ($n=1415$), (c) domestic livestock (cattle, sheep, goats, pigs and horses; $n=616$) and (d) domestic carnivores (dogs and cats; $n=374$). Fig. 1(b)–(d) were recreated with data from Cleaveland et al. (2001). For comparison with other host groups, 46 arthropod parasites from non-human primates were omitted.

could influence patterns of host specificity. First, taxonomic patterns pointed to a possible role for fast replication and high genetic variability in facilitating infection of multiple hosts. Second, links between transmission mode and host specificity indicated that transmission might determine encounter probabilities between host and parasite species.

Viruses might be dominated by generalist pathogens because their high mutation rates, greater antigenic diversity, high population diversity and short generation times (Morand et al., 1996; Whitlock, 1996; Gupta et al., 1998) allow them to rapidly adapt to a larger number of hosts. In fact, nearly half of primate viruses could infect hosts from multiple orders, and virtually all of these (38/39) multi-order viruses were RNA viruses, which have higher mutation rates than DNA viruses (Drake, 1991; Domingo and Holland, 1997; Holmes, 2003). This taxonomic bias in levels of host specificity is consistent with recent analyses of zoonotic pathogens and risk of emergence in humans and domesticated species, as previous studies showed that RNA viruses of humans are more than twice as likely to be zoonotic than DNA viruses (Cleaveland et al., 2001; Taylor et al., 2001).

Relative to viruses, protozoa were evenly distributed among the specificity classes, and nearly half of the helminths reported from primates were recorded as species-specific (Fig. 1(c)). Helminths might have longer generation times than most microparasites (Anderson and May, 1991), and their relatively more complex life history strategies could limit their ability to infect or adapt to new host species. However, the strong associations between specificity and parasite type could also be

Table 2
Characteristics of shared wild primate and human parasites (human records taken from Ashford and Crewe, 1998; Taylor et al., 2001)

(a) Parasite type

Parasite type	No. reported to infect wild primates and humans	No. recorded as emerging in humans
Arthropod	4	–[a]
Bacteria	19	12
Fungi	5	3
Helminth	33	4
Protozoa	24	6
Virus	30	26

(b) Transmission strategy

Transmission strategy	No. reported to infect wild primates and humans	No. recorded as emerging in humans
Close contact	13	9
Close + non-close	38	17
Non-close contact	16	4
Vector	30	19
Intermediate host	15	1

[a] Taylor et al. (2001) did not include arthropods.

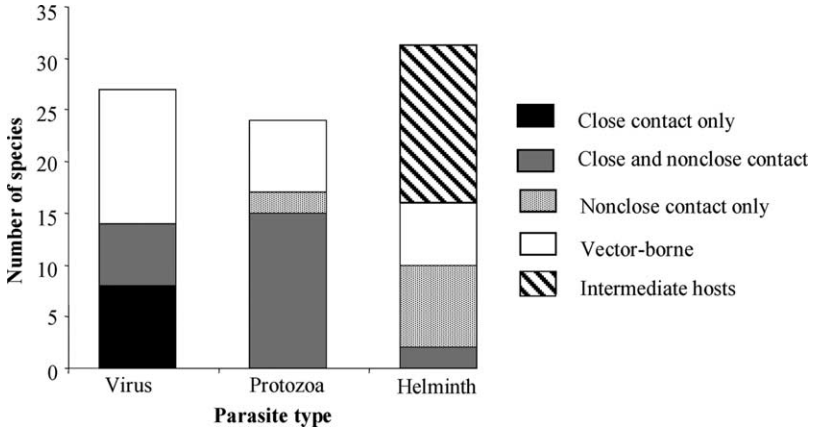

Fig. 5. Major transmission strategies of viruses ($n=30$), protozoa ($n=24$), and helminths ($n=33$) reported to infect both wild non-human primates and humans. Nearly all of these pathogens (>90%) were recorded as multi-order or multi-family generalists in the primate database.

influenced by taxonomic biases innate in applying the concept of a 'species' across a wide diversity of phyla. Because this bias is potentially substantial, we restricted most of our analyses to comparisons within the three major parasite groups.

Clear but contrasting patterns emerged between specificity and transmission type within viruses, protozoa and helminths. First, viruses were dominated by vector-borne pathogens capable of infecting hosts from multiple orders, and close contact (including sexual and vertical) transmitted pathogens with greater levels of host specificity (Fig. 3(a)). As predicted, most viruses capable of sexual transmission, including SIVs and STLVs, were highly host specific, although most of these were also capable of transmission by close contact and vertical routes. Second, most protozoan parasites fell into one of two transmission-specificity categories: multihost (multi-family or order) parasites transmitted by a combination of close and non-close contact, and vector-borne parasites that were more host specific (Fig. 3(b)). Helminths were by far the most host-specific primate parasites, but were also dominated by species transmitted by indirect routes, including non-close contact, vectors, and intermediate hosts (Fig. 3(c)). Specificity patterns among helminths infecting wild primates were difficult to characterise, possibly due to the diverse groups encompassed within helminths (i.e. acanthocephalens, nematodes, cestodes and trematodes). Nevertheless, these tended to fall into three major groups: highly specific parasites capable of transmission by combined close and non-close contact, vector-borne parasites with relatively high specificity, and parasites transmitted by intermediate hosts that included both highly specific and multi-order parasites (Fig. 3(c)).

Patterns reported from our data set have implications for understanding disease risk in humans and domesticated species, in part because wild primates are humans' closest relatives, and also because of a growing awareness that many emerging diseases are multihost pathogens that can be shared among humans, domesticated animals, and wildlife. Given their close evolutionary relationship, the taxonomic distribution of parasites from wild primates was surprisingly unlike patterns from humans (Taylor et al., 2001; Cleveland et al., 2001), which were dominated by bacteria and fungi (Fig. 4). Nevertheless, over 100 parasite species from wild primates were also reported in humans (Fig. 5), and nearly all of these could infect hosts from multiple orders or families. Moreover, 46 wild primate parasites were also reported as emerging in human populations, and nearly half of these were viruses transmitted by close contact or arthropod vectors. Further investigation should reveal which of these pathogens are associated with primates as reservoir hosts, and which are primarily human pathogens that spill over into wild primates (e.g. Haydon et al., 2002).

Although we believe this study provides an important first step in quantifying the characteristics of pathogens from wild host species, several limitations of this dataset should be noted. First and foremost, although we systematically searched for wild primate parasites, our list is unlikely to be complete, in part because many primate species have not been sampled adequately for parasites in the wild (Nunn et al., 2003). Similarly, our study might be limited by sampling biases towards particular pathogen groups and accurate knowledge of parasite taxonomy. Thus, one explanation for the high proportion of multi-host parasites among viruses is that virus taxonomy is poorly resolved. Improved information could lead to viruses currently classified as multihost being separated into a series of host-specific species (i.e. 'host races'). Moreover, biased sampling might occur for parasites among different taxonomic groups. For example, nematologists might be most interested in collecting and describing new helminth species, whereas virologists might be most interested in studying zoonotic pathogens. Although we attempted to control for study biases in parasite sampling effort by calculating specificity confidence scores and running the analyses excluding those with low confidence, analyses of confidence scores and citation counts suggest that well studied parasites have a wider reported host range.

Overall, our results indicate that there are fundamental differences in specificity within the taxonomic groups and that these differences are significantly related to transmission strategies, but these patterns might change as our understanding of parasite taxonomy and sampling effort improve.

The distribution and population biology of multi-host pathogens is becoming increasingly important for wildlife conservation and human health (Murray et al., 1999; Daszak et al., 2000), and adding multiple host species to infectious disease systems can have major consequences for disease spread and evolution (Frank, 1993; Begon et al., 1999; Woolhouse et al., 2001; Antonovics et al., 2002; Gandon, 2002; Holt et al., 2003; Gandon, 2004). For example, the presence of reservoir hosts can lead to periodic pathogen resurgence following long durations of disease-free periods in highly susceptible host species (Cleaveland and Dye, 1995; Keeling and Gilligan, 2000; Haydon et al., 2002; Swinton et al., 2002), and parasites in multiple host systems can intensify disease impacts on sensitive wildlife species (Greenman and Hudson, 2000). For other pathogens, such as *Borrelia burdorferi* (the causative agent of Lyme disease), a greater diversity of host species can reduce pathogen impacts and prevalence (Schmidt and Ostfeld, 2001; LoGiudice et al., 2003). The presence of multiple host species could also affect the evolution of pathogen virulence and the evolution of transmission routes among different hosts (Gandon, 2004).

In summary, despite the fact that parasites are often assumed to be under selection for specialisation on commonly infected host species (Fry, 1996; Whitlock, 1996; Kawecki, 1998), multihost pathogens are common in wild primate hosts. These multihost pathogens might experience several advantages, including reduced risks of ecological extinction, as their fates are not linked to the success of one or a few host species (Poulin, 1998). Results from our study provided limited support for two key processes that might favor the occurrence of generalism, including the ability to disperse to and infect multiple host species. However, our results also indicate that the evolutionary and ecological pressures influencing parasite transmission and host specificity differ among parasite taxonomic groups, perhaps because of their unique biology and life cycles. A clearer understanding of the limits to and opportunities for specialisation within each parasite group should help to explain these patterns, as will a clearer picture of potential sampling biases that could influence observed records of host and pathogen species combinations.

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